Dichlorvos (DDVP)

SECOND ADDENDUM[1]

to Risk Characterization Document

Medical Toxicology Branch

Department of Pesticide Regulation

California Environmental Protection Agency

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[1] Addendum to Risk Characterization Document for Dichlorvos (DDVP) (L.O.Lim, C.N.Aldous, S.R.Morris, J.F.Gee, H.R.Fong, T.A.Formoli, C.J.Rech, and K.F.Pfeifer, January 19, 1996) and first Addendum (November 4, 1997).

I. SUMMARY

INTRODUCTION

This second addendum reevaluated the risk assessment of dichlorvos (DDVP) because of new information on the toxicology and exposure of DDVP. The exposure scenarios assessed were: acute occupational and residential exposures, as well as chronic and lifetime dietary exposures.

RISK ASSESSMENT

Hazard identification

For acute occupational and residential exposures, a route-specific approach was considered. The critical no-observed-effect level (NOEL) used to assess dermal exposure was 0.5 mg/kg/day based on red blood cell cholinesterase inhibition in humans after oral dosing. The critical adjusted NOEL for inhalation exposure was 0.325 mg/kg/day for cholinergic signs and mortality in rabbits. In the 1996 Risk Characterization Document (RCD), the acute inhalation NOEL was used for the total exposure by both routes.

For lifetime exposures of all routes, the additional data submitted did not change the conclusion that there is sufficient evidence for DDVP oncogenicity.

Exposure

The occupational and residential exposures were determined for each route of exposure (inhalation and dermal) based on information given in the 1996 Risk Characterization Document.
The chronic dietary exposure was recalculated based on U.S. EPA analyses of the current monitoring data and field trials. Since the current data showed DDVP residues were essentially at the detection limit, the dietary exposure was substantially reduced.

CONCLUSIONS

This Addendum reassessed several acute, chronic, and lifetime exposure scenarios to DDVP. For warehouse workers and livestock applicators, the inhalation route of exposure was of concern as the margins of exposures (MOEs) were below the benchmark. For residents, both the inhalation and dermal exposures were of concern as the MOEs were below the benchmark for all uses (structural, fogger, and resin-strip), except pet collar. The chronic and lifetime dietary exposures to DDVP were no longer of concern. The exposure estimates were reduced because current data showed residue levels at the detection limit for almost all foods.

Summary of margins of exposure for occupational, residential, and dietary exposures to DDVP.

Scenarios	Acute exposure			Chronic exposure	Lifetime exposure	
	Inhalation MOE[a]	Dermal MOE[b]	Oral MOE[b]	MOE[c]	risk[d] (q1)	risk[d] (q1*)
Occupational						
Warehouse worker	**27**	**NA[e]**	**NA**	42	6×10^{-5}	1×10^{-4}
Structural PCO	**650**	**57**	**NA**	31	8×10^{-5}	1×10^{-4}
Livestock applicator	**6**	**71**	**NA**	5	5×10^{-4}	9×10^{-4}
Residential						
Structural use	**32**	**NA**	**NA**	125	2×10^{-5}	4×10^{-5}
Home-use fogger (child)	**10**	**9**	**455**	12	-	-
Home-use fogger (adult)	**10**	**6**	**30**	20	1×10^{-4}	2×10^{-4}
Pet owner	**1121**	**NA**	**NA**	500	6×10^{-6}	1×10^{-5}
Resin strip (child)	**8**	**NA**	**NA**	4	-	-
Resin strip (adult)					5×10^{-4}	9×10^{-4}
Dietary						
Population subgroups			303-1027	**>23000**	-	-
General population			568	**>50000**	**2×10^{-7}**	**3×10^{-7}**

Bolded values are those recalculated in this Addendum. Other values were those calculated in the 1996 Risk Characterization Document.

a Margin of exposure (MOE) was based on an adjusted inhalation NOEL of 0.325 mg/kg/day for cholinergic signs and mortality in rabbits. The benchmark for health concern is 100.

b MOE was based on an oral NOEL of 0.5 mg/kg/day for red blood cell cholinesterase inhibition in humans. The benchmark for health concern is 10.

c MOE was based on an adjusted inhalation NOEL of 0.025 mg/kg/day and oral NOEL of 0.05 mg/kg/day for both brain cholinesterase (ChE) inhibition in the rat (inhalation) and in the dog (oral), respectively. The benchmark for health concern is 100.

d Oncogenic risk was based on the human equivalent potency factors of 0.20 $mg/kg/day^{-1}$ and 0.35 $mg/kg/day^{-1}$ for q1 and q1*, respectively. The

benchmark for health concern is 1×10^{-6}.

e No dermal or oral exposure.

II. INTRODUCTION

In the 1996 DDVP Risk Characterization Document (RCD, Lim *et al.*, 1996) and the first Addendum (Lim, 1997) to the Risk Characterization Document, the following scenarios had margins of exposure or oncogenic risks of concern: (1) acute, chronic, and lifetime exposures of workers and residents, except pet collar users; (2) chronic and lifetime dietary exposure of the U.S. population. Furthermore, an additional uncertainty factor was considered not needed to address potential increased sensitivities of infants and children to DDVP as mandated by the Food Quality Protection Act. Some of these conclusions were reevaluated in this Addendum because new information on the toxicology and dietary exposure of DDVP was submitted to DPR.

The specific scenarios addressed in this Addendum are: (1) acute occupational and residential exposure using route-specific NOELs, and (2) chronic and lifetime dietary exposures using more recent residue data. The chronic and lifetime occupational and residential exposures were not reevaluated because there is no change in the NOEL or potency factors. These exposure levels will be affected by mitigation measures for acute exposure. The acute dietary exposure was not reevaluated because the MOEs were greater than 300 in the 1996 RCD. The use of current residue data would increase the MOE well above the benchmark of 100. The combined exposures were also not included in this Addendum since the major component is the acute occupational and residential exposures. The MOEs calculated previously and those in this Addendum for some of acute exposure scenarios were already below the benchmarks.

III. TOXICOLOGY PROFILE

A. Acute Toxicity and Neurotoxicity

Additional information on the acute toxicity of DDVP was recently submitted to DPR (as listed below). However, the results from these studies did not change the acute NOELs established in the RCD (Lim *et al*., 1996) and the first Addendum (Lim, 1997).

(1) A review of animal and human studies using slow-release strips (Arts, 1995).
(2) Aquatic acute toxicity studies of DDVP and 4-E emulsifiable concentrate conducted with sheepshead minnow (Jones and Davis, 1994a, b), Eastern oyster (Jones and Davis, 1994 c, 1995), mysid (Jones and Davis, 1994d, e).
(3) A report on the acute inhalation toxicity in swine (Kirkland, 1971).
(4) Supplemental reports on methodology for the human studies (Gledhill, 1997 a, b, c).
(5) A 28-day neurotoxicity in hen (Redgrave, 1994). A summary is provided in Appendix A.

B. CHRONIC TOXICITY AND ONCOGENICITY

In the RCD, DPR considered DDVP oncogenic by the oral route based on increased mononuclear cell leukemia (MCL) observed in rats given DDVP by gavage in an oncogenicity study (Chan, 1989). The result was also applied to the inhalation route as evidence of

oncogenicity since the only available inhalation study was considered inadequately conducted. The U.S. EPA considered DDVP to be oncogenic only by the oral route and not by the inhalation route. The basis for not quantifying the cancer risk by the inhalation route was that the 2-year inhalation study in rats did not show any increased tumor incidence (U.S. EPA, 1996). U.S. EPA classified DDVP as a Class C, possible, human carcinogen.

The DPR position on DDVP oncogenicity was reevaluated in this Addendum because of the following information:

(1) A Pathology Working Group for the registrant (Amvac Chemical Corporation) determined that there was no increase in the severity of MCL (progression from Stage 1 to other stages) with the dose (Brown, 1995; Manley, 1995a) (Table 1). The Working Group considered the increased incidence of MCL as equivocal evidence of a possible carcinogenic effect induced by DDVP.

(2) At the recent U.S. EPA FIFRA Scientific Advisory Panel (SAP), several members commented on the oncogenicity of DDVP (Lewis, 1998). They noted that forestomach tumors were likely to be due to irritation and should be evaluated by a threshold approach. As for MCL, the interpretation of the data was not so clear. MCL is a common tumor type with variable background rate and is specific to the Fisher rat. While the effect was treatment-related, a dose-response relationship was not demonstrated. In addition, the staging results for MCL by the Working Group did not showed increased severity. The final SAP report concluded that DDVP was a weak oncogen.

After a reexamination of the data, DPR found that the data from the oral chronic toxicity study clearly showed elevated incidences (two-fold higher than the control) after DDVP treatment (Chan, 1989). Since only one study has been conducted, dismissing the finding with the assumption that the result was part of the background incidence, is not appropriate. The DDVP treated groups also showed a higher number (10/50 rats) of animals in Stage 3 of MCL than that (5/50 rats) for the control. While the increase was not statistically significant, it could be used as evidence for potential oncogenicity.

The inhalation chronic toxicity study showed a significantly increased incidence of pituitary adenomas in the female rats (Blair *et al.*, 1974). In the 1996 RCD, DPR was concerned that tumors in the males may be masked by high incidences of autolyzed tissues. However, a reexamination of the data showed that there was a sufficient number of male rats with intact tissues to show no oncogenic effect (Table 2).

Table 1: Staging of mononuclear cell leukemia in rats after chronic exposure to DDVP[a].

Stage of MCL[b]	control	low dose	high dose
0	39/50 (78%)	30 (60%)	29 (58%)
1	4/50 (8%)	5 (10%)	2 (4%)
2	2/50 (4%)	5 (10%)	9 (18%)
3	5/50 (10%)	10 (20%)	10 (20%)

a Data from Brown, 1995. Incidences at the dose groups were number affected/number animals examined.

b Stage 0=no leukemia. The criteria for Stage 1 were that the spleen not enlarged or slightly enlarged with small numbers of neoplastic mononuclear cells; no or very few mononuclear cells in the liver, but not in other organs. The criteria for Stages 2 and 3 showed progressively increased in spleen enlargement, increased numbers of mononuclear cells, and involvement with other organs.

Table 2. Pituitary adenoma in rats after chronic inhalation exposure to DDVP[a].

DDVP(ppm)	Incidence of pituitary adenoma	
	males	females
0	4/31 (13%)	7/43++ (17%)
0.05	10/32 (31%)	5/44 (11%)
0.5	6/31 (19%)	12/39 (31%)
5	10/41(24%)	16/45* (36%)

a Data from Blair et al., 1974. ++ Significance at 0.01 by Peto's chi-square trend test. * Significance at 0.05 by Fisher's Exact Test. Incidences (number affected/number examined) are those in animals survived >49 weeks (exclude those with autolyzed tissues).

IV. RISK ASSESSMENT

A. HAZARD IDENTIFICATION

1. Acute Toxicity

For oral exposure, the critical NOEL determined in the 1996 RCD and the 1997 Addendum remained the same. For inhalation and dermal exposures, a route-specific approach was considered in this Addendum because of the following reasons: (1) a route-specific evaluation was needed to determine appropriate mitigation measures, and (2) U.S. EPA used the human oral studies to establish a NOEL in order to assess the dermal exposure (U.S. EPA, 1998). The critical NOEL used to assess dermal exposure was 0.5 mg/kg/day based on red blood cell cholinesterase inhibition in humans after oral exposure (Gledhill, 1977 d, e, and f). The critical NOEL for inhalation exposure remained the same at 0.325 mg/kg/day for cholinergic signs and mortality in rabbits (Thorpe *et al.*, 1971).

2. Oncogenicity

DPR remains concerned about the oncogenicity of DDVP by both the oral and inhalation routes. As presented in the 1996 RCD, oncogenicity studies showed that DDVP caused MCL in the male rat (gavage), pituitary adenomas in the female rat (inhalation), and forestomach tumors in the female mouse (gavage). The lack of statistically significant increase in severity of MCL does not negate the findings of increased incidence with DDVP treatment. Results from genotoxicity studies provided additional evidence for potential oncogenicity. DDVP was genotoxic in some *in vitro* systems, mouse lymphoma forward mutation assay, and unscheduled DNA synthesis assay using human epithelial cells. There was also evidence that DDVP interacts with DNA as methylated DNA was detected in tissues of mice given DDVP by intraperitoneal injection (Segerback, 1981). While DDVP was negative in some genotoxicity assays, these results only indicate that a positive response was not detected in the particular test systems under specific conditions. They do not negate the positive findings from oncogenicity and genotoxicity studies.

B. EXPOSURE ASSESSMENT

1. Occupational and Residential Exposures

The route-specific acute exposure levels (ADD) were determined from the Appendix B in the 1996 RCD (Table 5). There was no change to the estimates for chronic (AADD) or lifetime (LADD) exposures.

Table 5. Acute occupational and residential exposures by inhalation, dermal, and oral routes.

Scenarios	ADD[a] Route-specific daily exposure (*u*g/kg/day)		
	Inhalation	Dermal	Oral
Occupational			
Warehouse worker	11.93	0	0
Structural PCO	0.5	8.73	0
Livestock applicator	54.71	7.06	0
Residential			
Structural use	10.14	0	0
Home fogger (child)	31.5	57.2	1.1
Home fogger (adult)	33.3	84.6	16.6
Pet owner	0.29	0	0
Resin strip (child)	-	-	-
Resin strip (adult)	42.5	0	0

a Based on values provided in Appendix B of the 1996 RCD (Lim *et al.*, 1996).

2. Dietary Exposure

In the 1996 RCD, the residue levels were based on either the tolerance (meat products) or a market survey for processed commodities conducted by Amvac Chemical Corporation (Williams, 1991). DPR also considered residue data used by the U.S. EPA as part of harmonization effort between DPR and U.S. EPA. Recently submitted studies provided more evidence that DDVP residues were reduced during processing and dissipated with time (Appendix C). In addition, the U.S. EPA has revised the dietary exposure estimates based on more recent monitoring studies (FDA Total Diet Study and Monitoring Studies, USDA Pesticide Data Program) and registrant conducted field trials (Hummel, 1998a and b; Steinwand, 1998).

Since the chronic and lifetime dietary exposure estimates in the 1996 RCD resulted in margins of exposure or risks close to the benchmarks of health concern, these exposures were recalculated in this Addendum. As a continuing effort on harmonization, the residue values and percentage of crop treatment used were those determined by the U.S. EPA (Hummel, 1998a and b; Steinwand, 1998). Chronic dietary exposures were also based on the consumption rates from the 1989-1992 Continued Surveys of Food Intake of Individuals. The chronic exposure values were substantially lower than those in the 1996 RCD (0.06 -0.53 *u*g/kg/day) and were in the range of 0.000209 *u*g/kg/day (nursing infants) to 0.002124 *u*g/kg/day (nonnursing infants) (Appendix D). The highest chronic exposure for adults was 0.00078 *u*g/kg/day for females (13+ years old and nursing). The chronic exposure for the general population in the Pacific region was 0.000985 *u*g/kg/day.

3. Combined Exposure

The acute and chronic dietary exposures for DDVP are substantially reduced because current residue databases showed lower residue residues than those used in the RCD. Consequently, the combined exposure levels were essentially those from occupational or residential exposures alone.

C. RISK CHARACTERIZATION

The revised critical NOELs for acute exposure risk characterization are presented in Table 4. It should be noted that in the 1996 RCD, cholinesterase inhibition was used to calculate the MOE for lifetime exposure. Since lifetime exposure levels are only appropriate to address oncogenicity, any reference to the MOEs for long-term exposures should just be those calculated for chronic exposure only (under "Chronic Exposure" column in Table 23).

Table 4. The critical no-observed-effects levels (NOELs) and potency factors for risk characterization.

Scenarios	Routes of exposure	Adjusted [a] NOEL *u*g/kg/day	Effects/species	References[b]
Acute				
occupational residential combined	inhalation	325	death/rabbit (2-3 days)	Thorpe *et al.*, 1971
	dermal	500	erythrocyte ChE inhibition/human	Gledhill, 1997e
dietary	oral	500	erythrocyte ChE inhibition/human	Gledhill, 1997e
Chronic				
occupational residential combined	inhalation	25	brain ChE inhibition, ↓body weights/rat	Blair *et al.*, 1974
dietary	oral	50	brain ChE inhibition/dog	Markiewicz, 1990*
	Potency factors	human equivalent mg/kg-day^{-1}		
Lifetime	oral inhalation	q_1=0.20 q_1*=0.35	mononuclear leukemia/rat	Chan, 1989*

a/ Inhalation NOELs in mg/m^3 were adjusted by converting doses to mg/kg-day units using equations in Appendix D (1996 RCD) and corrected for an absorption factor (50%). The oral absorption was assumed to be 100%.

b/ * indicates study was acceptable to DPR according to FIFRA guidelines.

1. Occupational Exposure (Tables 6 and 7)

For the workers, the MOEs for acute inhalation exposures were 6, 27, and 650 for livestock applicators, warehouse workers, and structural pest control operators (PCO), respectively. The MOEs for acute dermal exposure were 57 and 71 for structural PCOs and livestock applicators. The change to route-specific NOELs had an impact only for structural PCOs. Since the primary route of exposure for the PCO was dermal, the MOE for the inhalation route was much higher (650) than that (57) for the dermal route. The MOE was 36 for total exposure determined in the 1996 RCD.

2. Residential Exposure (Tables 6 and 7)

For residents, the MOEs for inhalation exposure ranged from 8 (child exposed to resin strips) to 1121 (pet owner). The MOEs for dermal exposure to foggers were 6 and 9 for adults and children, respectively. The MOEs for oral exposure to foggers were 30 and 455 for adults and children, respectively. The use of route-specific NOEL had little impact on the MOEs for these groups and are similar to those calculated based on total exposure for both routes since the primary exposure was inhalation.

Table 6. Margins of exposure for occupational and residential acute exposures.

Scenarios	Total exposure	Route-specific exposure		
	MOE[a] (1996 RCD)	Inhalation MOE[a]	Dermal MOE[b]	Oral MOE[b]
Occupational				
Warehouse worker	27	27	NA[c]	NA
Structural PCO	36	650	57	NA
Livestock applicator	5	6	71	NA
Residential				
Structural use	33	32	NA	NA
Home fogger (child)	3	10	9	455
Home fogger (adult)	4	10	6	30
Pet owner	1083	1121	NA	NA
Resin strip (child)	8	8	NA	NA
Resin strip (adult)	-	-	-	-

a MOE was based on an acute inhalation adjusted NOEL of 0.325 mg/kg/day for cholinergic signs and mortality in rabbits (Thorpe *et al.*, 1971).

b MOE was based on an acute oral NOEL of 0.5 mg/kg/day for RBC cholinesterase inhibition in humans (Gledhill, 1997e).

c No dermal or oral exposure.

3. Dietary Exposure (Table 7)

The margins of exposure for chronic dietary exposure increased to > 23000 (Appendix D) compared with those (95 to 861) determined in the 1996 RCD. The oncogenic risks decreased to 2×10^{-7} and 3×10^{-7}, for q1 and q1*, respectively. The risks in the 1996 RCD were 7×10^{-6} and 3×10^{-5}, for q1 and q1*, respectively.

Table 7. Margins of exposure and oncogenic risks for occupational, residential and dietary chronic and lifetime exposures.

Scenarios	Chronic exposure	Lifetime exposure	
	MOE[a]	risk [b] (q1)	risk[b] (q1*)
Occupational			
Warehouse worker	42	6×10^{-5}	1×10^{-4}
Structural PCO	31	8×10^{-5}	1×10^{-4}
Livestock applicator	5	5×10^{-4}	9×10^{-4}
Residential			
Structural use	125	2×10^{-5}	4×10^{-5}
Home fogger (child)	12	-	-
Home fogger (adult)	20	1×10^{-4}	2×10^{-4}
Pet owner	500	6×10^{-4}	1×10^{-5}
Resin strip (child)	4	-	-
Resin strip (adult)	-	5×10^{-4}	9×10^{-4}
Dietary			
Population subgroups	>23000		
Pacific Region	>50000	2×10^{-7}	3×10^{-7}

a MOE was based on an adjusted inhalation NOEL of 0.025 mg/kg/day and an oral NOEL of 0.05 mg/kg/day for brain ChE inhibition in the rat (Blair *et al*., 1974) and in the dog (Markiewicz, 1990).

b Oncogenic risk based on the human equivalent potency factors of 0.20 $mg/kg/day^{-1}$ and 0.35 $mg/kg/day^{-1}$ for q1 and q1*, respectively (Chan, 1989).

IV. RISK APPRAISAL

The dose (the no-observed-effect level or NOEL) at which adverse effects did not occur was used to assess the non-cancer hazard for potential one-time and long-term exposure to humans. For cancer effects, the potency factors were used. The margin of exposure (MOE, the quotient of NOEL/exposure) and risks were compared with the conventional benchmark levels considered protective of human health. For the MOE approach, the benchmark MOEs are 100 and 10 for data from animal and human studies, respectively. For oncogenic effects, the benchmark risk levels were equal to or less than 1×10^{-6}. Review of the database did not support the proposal by Amvac Chemical Corporation that a MOE of 10 was sufficient for all exposure durations (Wilkinson, 1995; Stonard, 1997).

A. Occupational, Residential, and Dietary Exposures

The acute inhalation MOEs were less than 100 for warehouse workers, livestock applicator, and residents from home-uses, foggers, and resin-strips. The inhalation MOEs of structural PCOs and pet owners were greater than 100. The acute dermal MOEs were less than 10 for residents expose to DDVP in foggers. For occupational uses, the dermal MOEs were greater than 10. For both occupational and residential exposures, the oncogenic risk did not meet the standard benchmarks for acceptable risks.

The chronic dietary exposure of the general population in the Pacific region and other subgroups were greater than 100. The lifetime oncogenic risks were $2x\ 10^{-7}$ and 3×10^{-7}, for q1 and q1*, respectively. Amvac estimated risk was 1.2×10^{-7} (Smith et al., 1995). This risk level was considered conservative since it did not include an average 0.084 residue reduction factor for the covering (or removal) of food/feed commodities prior to the fogging of food handling establishments with DDVP.

B. Food Quality Protection Act Issues

U.S. EPA has proposed an additional uncertainty factor to address potential increased sensitivity of infants and children to DDVP because decreased brain weights in guinea pig pups reported in a published article (Mehl et al., 1994). DPR reviewed the study and identified the effect as a possible adverse effect. The deficiencies in the study included a single dam per dosing regimen and missing details of methodology.

The U.S. EPA Scientific Advisory Panel recently considered the study and the need for the uncertainty factor (Lewis, 1998). They concluded that such a factor, either 10-fold or 3-fold was needed because of the absence of developmental neurotoxicity studies, paucity of data on aggregate exposure, and potential cumulative exposure from other organophosphates. Amvac Chemical Corporation plans to conduct a similar study with a modified protocol.

V. CONCLUSIONS

This Addendum reassessed several acute, chronic, and lifetime exposure scenarios to DDVP. For warehouse workers and livestock applicators, the inhalation route of exposure was of concern as the margins of exposures (MOE) were below the benchmark. For residents, both the inhalation and dermal exposures were of concern as the MOEs were below the benchmark for all uses (structural, fogger, and resin-strip), except pet collars. The chronic and lifetime dietary exposures to DDVP were no longer of concern. The exposure estimates were reduced because current data showed residue levels at the detection limit for almost all foods.

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APPENDIX A- Neurotoxicity Study in Hens

DDVP (97.87% pure, 0, 0.3, 1.0, 3.0 mg/kg) were given by gavage to adult female domestic hens (21 hens/dose) for 28 days (Redgrave, 1994; Jortner, 1994; Manley, 1995b; Manley, 1992). Another group (3 hens) received 0.1 mg/kg DDVP for brain ChE activity determination at day 30. TOCP (7.5 mg/kg) and corn oil were positive control and vehicle control, respectively. The hens were observed for a total of 49 or 77 days after onset of dosing. Death occurred at 1.0 mg/kg (1/21 hens) and 3.0 mg/kg (4/21 hens). At 3.0 mg/kg, clinical signs of neurotoxicity included: wings outstretched, limping, inability to stand, quiet/subdued, unsteadiness. Unsteady gait and inability to stand was also observed at 1.0 mg/kg (2/21 hens). Axonal degeneration in the cerebellum, spinal cord, sciatic nerve and tibial nerve was increased at all doses.

Histopathology showed splitting/thickened and /or densely staining material within the myelin at all doses. The neurotoxicity NOEL was < 0.3 mg/kg. On day 4, brain ChE activities were 56% and 37% of control for the 1.0 mg/kg and 3.0 mg/kg groups, respectively. By day 30, brain ChE activities were 74%, 66%, and 46% of control, for 0.3 mg/kg, 1.0 mg/kg and at 3.0 mg/kg, respectively. The NOELs for brain ChE were 0.3 mg/kg and < 0.3 mg/kg for 4 and 28 days of exposure. This study was considered acceptable by DPR according to FIFRA guidelines.

The registrant submitted a peer review of this study by the Pathology Working Group in response to the DPR review (Hardisty, 1998). The Working Group did not consider the axonal degeneration to be treatment related and this review is pending evaluation by DPR. Since the study is not used for risk assessment, the DPR evaluation will have no impact on the conclusions of this Addendum.

APPENDIX B: Mechanism for Forestomach Tumors

The registrant submitted studies to address the mechanism for DDVP-induced forestomach tumors. These studies are pending review by the DPR. Since DPR did not use the forestomach tumor as an endpoint to calculated the potency for oncogenicity, the result of the review will unlikely have any impact on the risk assessment. A summary of the submitted studies is provided in this Appendix.

B6C3F1 mice (5/sex/group) were exposed to a single dose of DDVP (0, 10, 20, 40 or 100 mg/kg) by gavage (Benford, 1991a; Bremmer, 1993). The positive control groups received either N-methyl-N'-nitro-N-nitrosoguanidine (MNNG, a forestomach carcinogen) or butylated hydroxyanisole (BHA, a non-genotoxic promoter of forestomach tumors) (Benford, 1991b). After specified time period, unscheduled DNA synthesis, replicative DNA synthesis, and histopathology were determined in forestomach sections (Pearson, 1992; Benford, 1990). Results showed that DDVP, similar to BHT, caused focal hyperplasia and induced replicative DNA synthesis (Benford, 1992), but not unscheduled DNA synthesis. Therefore, authors concluded that the mechanism for DDVP-induced forestomach tumor was likely to be due to a non-genotoxic mechanism.

APPENDIX C: Residue Studies

The following are summaries of studies submitted by Amvac Chemical Corporation to be considered for dietary exposure assessment.

Hens (10-12/group) were fed DDVP (0, 2, 6, or 20 ppm) in capsules for 42 days to determine whether DDVP residues would occur when hens are feed DDVP treated bulk/packaged commodities processed into animal feed (March *et al.*, 1993). No residues (<0.01 ppm) were detected in the eggs or tissues (breast and thigh muscle, liver, kidneys, and fat).

DDVP residues were determined in peanuts stored in a warehouse which had been treated 88 times with DDVP (0.5 g ai/1000ft^3) in a three-month period (Hofen and Warnke, 1993a). The mean residue level in the whole peanuts was 36 ppm. These peanuts were then treated with a single application of DDVP (23.6 g ai/1000ft^3) before processing. DDVP residues were lower in the processing fractions than whole peanuts: 0.97 ppm in peanut meat before processing, <0.01 ppm in crude oil, refine oil, and soapstock, and 0.70 ppm in the pressed, solvent-extracted meal.

In another experiment, DDVP residues were determined at different depths (Hofen and Warnke, 1993c; Matheson, 1993). DDVP (0.5 g ai/1000ft^3) were applied above peanut pile daily for 9 months. Surface samples (0-3 inches) were collected monthly while subsurface samples (6, 12, 18, and 36 inches deep) were collected 1 (not 36 inches deep), 3, 5, 7, and 9 months. The highest residue levels for each depth and duration were: 46 ppm (surface, 4 months), 3.47 ppm (6 inches, 7 months), 0.44 ppm (12 inches, 5 months), 0.20 ppm (18 inches, 7 months), and <0.01 ppm (36 inches for all sampling period). DDVP residues were < 0.01 ppm (1 month samples) to 2.73 ppm (5 months) in the nutmeat of surface samples.

DDVP residues were also determined in field corn, wheat, rice, cottonseed, and soybean fractions before and after treatment with a single application of DDVP (23.6 g ai/1000ft^3 which is 10x usual rate) (Hofen and Warnke, 1993b). The results are summarized in the Table C1 and showed that DDVP residues were reduced in the processed products.

Table C1: Residue levels (in ppm) in grains stored in DDVP treated warehouses and grain fractions during processing [a].

fractions	field corn wet milled	field corn dry milled	wheat	rice	cottonseed	soybean
untreated all fractions	<0.01-0.02	<0.01-0.02	<0.0105-0.016	<0.0105-0.011	<0.0105-0.026	<0.01
whole after application	2.74	2.98	15.64	4.26	60.04	16.1
crude oil	0.03	3.68			15.13	9.90
refined oil	<0.01	0.41			1.23	<0.01
starch	<0.01		5.00 (shorts)	14.16 (hulls)	27.73 (hulls)	55.1 (hulls)
grits		1.58	15.05 (bran)	1.88 (bran)		
meal		0.63			0.45	0.37
coarse meal		1.65	4.27 (middlings)	0.023 (polished rice)	<0.0105 (soapstock)	<0.01 (soapstock)
flour		1.74	1.81			
grain dust	17.8		19.81	16.73		
reclaimed hexane	<0.01	0.03			0.089	0.08

a Data from Hofen and Warnke, 1993b.

DDVP residues in ten bulk-stored commodities were studied after a single application of DDVP (2 g ai/1000ft^3) (Schofield, 1993a). The commodities were stored in either open or covered (with plastic sheeting) tote bins during the application of DDVP to the warehouse by a fogger. The samples were collected about 6 hours after the application. Pretreatment residue levels were all below the detection limit of 0.01ppm. The residue levels are presented in Table C2. An additional experiment was conducted with samples collected 6, 12, 24, 48, 90, and 168 hours following the application to the open bins to determine the half-lives of some of these commodities under similar conditions (Schofield, 1993b). DDVP dissipation was most rapid with sugar (26 hours) and slowest with field corn (456-1000 hours). There was no correlation between residues and time for the walnut and peanut samples.

Table C2: DDVP residue levels in bulk-stored commodities after a single application[b].

Commodity	after application- highest reported residue (ppm)		half-life	regression
	open bin	covered bin		
cocoa bean	0.02	<0.01		
coffee bean	1.85	0.02		
dried bean	0.86	not determined	83 hours	0.8
field corn	0.84	<0.10	1000 hours 456 hours (without 6 and 48 hour samples)	0.026 0.486
flour	0.50	0.02	4-12 days	0.48
oat	0.99	not determined	278 hours 162 hours (without 12 and 24 hour samples)	0.149 0.756
soybean	0.44	<0.01	183 hours	0.663
sugar	0.41	<0.01	26 hours	0.982
tree nut	<0.01	not determined	no correlation for either walnut or peanuts	
wheat	0.30	not determined		

b Data from Schofield, 1993 a and b.

In a similar experiment, DDVP residues in packaged and bagged commodities were determined at pretreatment, 6 hours after each of 3 weekly applications, immediately before the 4th appli-cation, after the 4th application (Schofield, 1993h) (Tale C3). These samples were collected from the uppermost (top layer) portion of a pallet. Samples from the side and interior of the pallet were collected after the 4th application. To determine the half-life of residues, samples were collected at 6 to168 hours after the 4th application for selected commodities.

Table C3: DDVP residues in warehouse stored packaged and bagged commodities[a].

commodities	pretx (ppm)	Posttreatment (ppm)							
		1st appl.	2nd appl.	3rd appl.	before 4th appl.	after 4th application			half-life[b] (hrs)
						top	side	interior	
cereal	<0.01	<0.01	<0.01	<0.01	<0.01, 0.02	<0.01, 0.01	0.02, <0.01	<0.01, ND	
cocoa bean	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	
coffee bean	<0.01	0.06, 0.09	0.41, 0.02	0.14, 0.16	0.16, 0.21	0.18, 0.68	0.11, 0.06	0.03, <0.01	
cookie	<0.01	<0.01	<0.01, 0.03	<0.01	0.07, 0.04	0.03, <0.01	0.01, <0.01	<0.01, ND	
cracker	<0.01	<0.01	<0.01, 0.03	0.07, 0.03	0.33, 0.10	0.30, 0.03	0.32, 0.26	<0.01	
dried bean	<0.01	0.02, <0.01	0.05, 0.05	0.04, 0.03	0.06, 0.06	0.07, 0.16	0.08, 0.16	<0.01	139
field corn	<0.01	0.07, 0.09	0.35, 0.32	0.54, 0.78	0.43, 0.50	0.60, 0.58	<0.01, 0.15	<0.01	no[c]
flour	<0.01	0.07, 0.04	0.21, 0.62	0.57 0.32	0.46, 0.16	0.14, 0.12	0.16, 0.13	<0.01	314
oat	<0.01	0.03, <0.01	0.23, 0.25	0.37, 0.39	0.38, 0.49	0.69, 0.61	0.02, 0.17	0.02, <0.01	no
peanut	<0.01	0.38, 0.49	1.05, 0.29	0.52, 0.69	8.43, 1.45	0.97, 1.48	0.08, 6.89	3.84, 0.32	no
peanut nutmeat	<0.01					0.02, 0.05	<0.01, 0.55	0.28, 0.03	
soybean	<0.01	<0.01	0.03, 0.01	0.06, 0.05	0.12, 0.04	0.16, 0.10	0.04, 0.10	<0.01, 0.02	no
sugar	<0.01	0.05, <0.01	0.40, <0.01	0.04, 0.02	0.04, 0.02	0.05, 0.02	0.03, 0.03	0.01, 0.03	44
tree nut	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	no

a Data from Schofield, 1993h and 1993e.
b Assumes first-order kinetics and based on residue levels from 6 to 96 hours after application. No=
c no or poor correlation when first -order kinetics are assumed

A residue study of DDVP in animal feed products as a result of the use of DDVP (2 g ai/1000 ft^3) in an oat processing facility was conducted (Schofield, 1993c). Samples were collected prior to application, and after 1,3, and 6 turnover periods following application. Except for the fine groats samples, residues in all other fractions decreased with time (Table C4).

Table C4: DDVP residue levels in oat fractions after a single application to a processing facility[a].

Fractions	Pretreatment (ppm)	Posttreatment (ppm)[b]		
		T1	T3	T6
uncleaned oats	<0.01	<0.01	<0.01	<0.01
cleaned oats	<0.01	0.16, 0.63	0.03, 0.04	0.04, 0.14
fine groats	0.05, 0.06	2.47, 2.17	3.68, 6.08	8.10, 3.30
kiln outlet groats	<0.01	0.63, 0.36	0.16, 0.14	0.09, 0.08
oat hulls	<0.01	1.34, 0.60	0.47, 0.36	0.35, 0.43
steel cuts	<0.01	1.01, 0.95	0.43, 0.49	0.53, 0.54
Residue levels in steel cuts samples in different locations				
locations	6 hours	9 hours	12 hours	
31	4.20, 4.13	0.38, 0.43	0.42, 0.38	
32	6.6, 10.4	0.16, 0.19	0.12, 0.08	
34	4.13, 2.85	0.42, 0.79	0.53, 0.48	

a Data from Schofield, 1993c.
b T=turnover, defined as the time it takes for a commodity to travel during processing from the point of initiation to collection.

In another study, DDVP residues in oat products were determined after a single application (2 g ai/1000ft^3) in a manufacturing facility (Schofield, 1993d). As shown in the previous study, there was an increase in the residues in the fine groats while a decrease for the other fractions. The high residues in the flaked oats was not considered of human concern since the collected fraction routinely used for animal feed and not for human consumption. Monitoring of flaked oats at different areas of the facility showed a reduction of residues with time. A summary of the results is presented in the following table (Table C5).

Table C5: DDVP residue levels in oat fractions after a single application to a manufacturing facility[a].

Fractions	Pretreatment (ppm)	Posttreatment (ppm)[b]		
steel cut (storage)	<0.01			
steel cut (steamer)	<0.01	0.42, 0.07 (C1)[b]	0.02, 0.03 (C2)	0.03, 0.03 (C3)
fine groats	<0.01, 0.02	0.14, 0.10 (ES1)[c]	12.2, 8.63 (ES2)	
flaked oats	<0.01	0.97, 0.86 (T1)[d]	0.37, 0.37 (T3)	0.42, 0.37 (T6)
Residue levels in oat flak samples in different locations				
locations	6 hours	9 hours	12 hours	
22	4.65, 3.19	1.12, 2.14		
33	16.1, 5.63	0.60, 0.87	0.80, 1.16	
25	19.6, 28.9	1.59, 1.16	1.29, 0.80	

a Data from Schofield, 1993d.
b C1 sample was collected after the first flush of the commodity after equipment startup. C2 was collected 30 minutes after C1. C3 was collected 75 minutes after C1.
c ES1 sample was collected during the first flush of flaked oats through the system after equipment startup. ES2 was collected an interval when sufficient material has accumulated.
d T1 sample was collected after one turnover of the commodity equipment startup. T3 was collected after 3 turnovers. T6 was collected after 6 turnovers.

In a follow-up study on residues in oat processed products, the high DDVP residues in the fine groats were investigated (Feiler, 1993). Samples of the fines streams were collected over 16 hours following a single application of DDVP. The residue levels declined with time with the maximum residue levels ranged from 1.58 ppm (after application) to 0.56 ppm (16 hours post-application) (Table C6). While these levels exceeded the tolerance level of 0.5 ppm, the residue levels in the final feed in the truck samples ranged from 0.10 to 0.44 ppm.

DDVP residues in corn were also determined after a single application of DDVP (2 g ai/1000ft^3) in a corn processing facility (Schofield, 1993f). As shown in Table A6, the residues decreased with processing and with time.

Table C6: DDVP residue levels in corn fractions after a single application to a processing facility[a].

Fractions	Pretreatment (ppm)	Posttreatment (ppm)[b] T1	T2	T3
whole corn	<0.01	0.01, <0.01	0.01, 0.01	<0.01,<0.01
ground corn	<0.01	0.14, 0.06	0.04, 0.02	0.02, 0.02
ground corn (storage)	<0.01			<0.01, <0.01
Residue levels in ground corn samples in different locations				
locations	6 hours	9 hours	12 hours	
1	3.00, 1.93	0.29, 0.25	0.11, 0.18	
2	0.28, 0.45	0.01, 0.02	0.01, 0.01	
3	0.77, 0.89	0.15, 0.16	0.11, 0.05	

a Data from Schofield, 1993f.
b T1 sample was collected after one turnover of the commodity equipment startup. T3 was collected after 3 turnovers. T6 was collected after 6 turnovers.

The effect of processing (by heat) was studied on several raw agricultural commodities (Williams, 1993). The commodities (raw cocoa beans, dried pinto beans, tomato juice, coffee beans, raw hamburger meat, raw eggs, and raw whole milk) were fortified with known levels of DDVP and then processed using conventional commercial/home methods. The difference between the fortified level and the DDVP residue level recovered was considered as the loss from processing. A summary of the result is shown in Table C7. There was a correlation between the temperature and the loss from processing. Another possible factor is the fat content of the commodities since DDVP is liposoluble. This may account for the higher amount of DDVP residues retained in whole milk after pasteurization compared to other cooked commodities.

Table C7. DDVP residues in raw and cooked commodities[a].

precursor commodity	processing technique	final product	temper-ature (^{o}C)	time (minutes)	% loss from processing
raw cocoa beans	roast	chocolate liquor	135	10	99+
dried pinto beans	cook in water	cooked beans and liquor	>95	90	99-99+
tomato juice	condense	tomato paste	~80	40	90
ground, roasted coffee beans	brew with hot water	coffee	~100	8	66-75
raw hamburger meat	fry	cooked hamburger patties	>100	6	65-75
raw eggs	fry	scrambled eggs	>100	3	38
raw whole milk	pasteuri-zation	pasteur-ized whole milk	62.8	30	1.9-13

a Data from Williams, 1993.

DDVP residues were also determined in wheat fractions after a single application of DDVP (2 g ai/1000ft^3) in a wheat manufacturing facility (Schofield, 1993 g). Samples were collected after the first and third batch of production after DDVP application to the facility. A summary of the results is presented in Table C8.

Table C8. DDVP residues in wheat products after a single application in a wheat manufacturing facility[a].

Fractions	Pretreatment (ppm)	Posttreatment (ppm)	
		first batch	third batch
flour	<0.01	0.075, 0.055	0.033, 0.027
sugar	<0.10	0.094, 0.069	0.022, 0.027
dried milk	<0.01, ND	0.062, 0.047	0.10, 0.073
dried eggs	ND, <0.01	0.034, 0.039	0.093, 0.12
shortening (tank)	ND, ND	<0.032	<0.032
shortening (mixer)	<0.03, ND	<0.032, 0.034	0.036, <0.032
blended mix (mixer)		0.38, 0.30	0.19, 0.17
blended mix (tram)		<0.01, <0.01	<0.01, <0.01
blended mix (packaged)	<0.01, <0.01	A 0.17, 0.13 B 0.017, 0.021	A 0.019, 0.019 B 0.014, 0.014
DDVP residues in blended mix samples in different locations of the facility after application at various times (hours)			
locations	6.5 hours	9 hours	12 hours
1	5.42, 4.99	1.15, 1.13	0.67, 0.60
2	9.63, 7.75	0.56, 0.59	0.22, 0.17
3	4.61, 4.66	1.13, 1.18	0.42, 0.35

a Data from Schofield, 1993g.

APPENDIX D- Chronic Dietary Analysis

Chronic Exposure (EX1) Analysis for Dichlorvos
RESIDUE FILE NAME: DDVP1PCT ANALYSIS DATE: 7-14-1998
NFCS Combined 89-92 DATA
DPR NOEL (Chronic) = 0.050000 mg/kg body-wt/day
COMMENT 1: All uses based on USEPA DRES (6/11/98)
COMMENT 2: USEPA residue and percent crop treated as factor #2 adjustment

RESIDUE FILE LISTING

TAS CODE	CROP GRP	FOOD NAME	RESIDUE (PPM)	ADJ. FCTRS #1	#2	SOURCE CODE
14	N	GRAPES-RAISINS	0.000500	1.00	0.06	TDS
40	R	ALMONDS	0.003800	1.00	0.06	FTtr
41	R	BRAZIL NUTS	0.003800	1.00	0.06	FTtr
42	R	CASHEWS	0.003800	1.00	0.06	FTtr
43	R	CHESTNUTS	0.003800	1.00	0.06	FTtr
44	R	FILBERTS (HAZELNUTS)	0.003800	1.00	0.06	FTtr
45	R	HICKORY NUTS	0.003800	1.00	0.06	FTtr
46	R	MACADAMIA NUTS (BUSH NUTS)	0.003800	1.00	0.06	FTtr
47	R	PECANS	0.003800	1.00	0.06	FTtr
48	R	WALNUTS	0.003800	1.00	0.06	FTtr
49	R	BUTTER NUTS	0.003800	1.00	0.06	FTtr
50	A	PISTACHIO NUTS	0.003800	1.00	0.06	FTtr
51	R	BEECHNUTS	0.003800	1.00	0.06	FTtr
53	L	APPLES-DRIED	0.100000	1.00	0.06	FTtr
57	L	PEARS-DRIED	0.100000	1.00	0.06	FTtr
60	M	APRICOTS-DRIED	0.100000	1.00	0.06	FTtr
62	M	CHERRIES-DRIED	0.100000	1.00	0.06	FTtr
66	M	PEACHES-DRIED	0.100000	1.00	0.06	FTtr
68	M	PLUMS-PRUNES(DRIED)	0.100000	1.00	0.06	FTtr
73	A	BANANAS-DRIED	0.001800	1.00	0.06	FTtr
74	A	COCONUT	0.090000	1.00	0.06	FT
75	A	COCONUT-COPRA	0.090000	1.00	0.06	FT
77	A	DATES	0.000500	1.00	0.06	TDStr
78	A	FIGS	0.000500	1.00	0.06	TDStr
85	A	PAPAYAS-DRIED	0.001800	1.00	0.06	FTtr
90	A	PINEAPPLES-DRIED	0.001800	1.00	0.06	FTtr
96	A	LYCHEE-DRIED	2.000000	1.00	0.06	tolerance
110	A	COCOA BUTTER	0.000140	1.00	0.06	FT
111	A	CHOCOLATE	0.000140	1.00	0.06	FT
112	A	COFFEE	0.000500	1.00	0.06	TDS
113	A	TEA	0.000500	1.00	0.06	TDS
114	B	CHICORY	0.500000	1.00	0.06	PDPtr
115	S	ANISE	0.100000	1.00	0.06	FT
116	S	BASIL	0.100000	1.00	0.06	FT
117	S	CARAWAY	0.100000	1.00	0.06	FT
119	S	CINNAMON	0.100000	1.00	0.06	FT
121	S	CORIANDER	0.100000	1.00	0.06	FT
122	S	CUMIN	0.100000	1.00	0.06	FT
123	S	DILL	0.100000	1.00	0.06	FT
124	B	GINGER	0.100000	1.00	0.06	FT
126	B	HORSERADISH	0.100000	1.00	0.06	FT

FT=Field trial, tr=transferred (surrogate), PDP= Pesticide Data Program, TDS= Total Diet Study, FCTRS #2= (percentage of crop treated adjustment)/100.

127	S	ROSEMARY	0.100000	1.00	0.06	FT
128	S	MARJORAM	0.100000	1.00	0.06	FT
129	S	OREGANO	0.100000	1.00	0.06	FT
130	A	MUSTARD SEED	0.050000	1.00	0.06	FT
131	A	NUTMEG	0.100000	1.00	0.06	FT
133	S	SAGE	0.100000	1.00	0.06	FT
134	S	SAVORY	0.100000	1.00	0.06	FT
135	S	BAY	0.100000	1.00	0.06	FT
136	S	THYME	0.100000	1.00	0.06	FT
137	A	TURMERIC	0.100000	1.00	0.06	FT
138	A	ALLSPICE	0.100000	1.00	0.06	FT
179	S	FENNEL	0.100000	1.00	0.06	FT
200	S	CHIVES	0.140000	1.00	0.06	FTtr
206	D	ONIONS-DEHYDRATED OR DRIED	0.500000	1.00	0.06	FT
210	B	POTATOES(WHITE)-DRY	0.007500	1.00	0.06	FT
227	G	BEANS-DRY-GREAT NORTHERN	0.000500	1.00	0.06	TDS
228	G	BEANS-DRY-KIDNEY	0.000500	1.00	0.06	TDS
229	G	BEANS-DRY-LIMA	0.000500	1.00	0.06	TDS
230	G	BEANS-DRY-NAVY (PEA)	0.000500	1.00	0.06	TDS
231	G	BEANS-DRY-OTHER	0.000500	1.00	0.06	TDS
232	G	BEANS-DRY-PINTO	0.000500	1.00	0.06	TDS
237	O	CORN/POP	0.000500	1.00	0.06	TDS
239	A	PEANUTS-WHOLE	0.075000	1.00	0.06	FT
240	G	PEAS (GARDEN)-DRY	0.000500	1.00	0.06	TDS
242	G	LENTILS-WHOLE	0.000500	1.00	0.06	TDS
243	G	LENTILS-SPLIT	0.000500	1.00	0.06	TDS
246	A	SUNFLOWER-SEEDS-WITH HULLS	0.090000	1.00	0.06	FTtr
247	G	CAROB	0.000190	1.00	0.06	FT
249	G	BEANS-DRY-BROADBEANS	0.000500	1.00	0.06	TDS
251	G	BEANS-DRY-PIGEON BEANS	0.000500	1.00	0.06	TDS
252	A	SESAME SEEDS	0.090000	1.00	0.06	FTtr
254	A	PINENUTS	2.000000	1.00	0.06	tolerance
256	G	BEANS-DRY-HYACINTH	0.000500	1.00	0.06	TDS
258	G	BEANS-DRY-BLACKEYE PEAS/COWPEA	0.000500	1.00	0.06	TDS
259	G	BEANS-DRY-GARBANZO/CHICK PEA	0.000500	1.00	0.06	TDS
261	A	MUSHROOMS	0.003800	1.00	0.15	FT
265	O	BARLEY	0.000500	1.00	0.06	TDS
266	O	CORN/GRAIN-ENDOSPERM	0.000500	1.00	0.06	TDS
267	O	CORN/GRAIN-BRAN	0.000500	1.00	0.06	TDS
268	O	CORN SUGAR	0.000500	1.00	0.06	TDS
269	O	OATS	0.000500	1.00	0.06	TDS
270	O	RICE-ROUGH (BROWN)	0.000500	1.00	0.06	TDS
271	O	RICE-MILLED (WHITE)	0.000500	1.00	0.06	TDS
272	O	RYE-ROUGH	0.000500	1.00	0.06	TDS
273	O	RYE-GERM	0.000500	1.00	0.06	TDS
274	O	RYE-FLOUR	0.000500	1.00	0.06	TDS
275	O	SORGHUM (INCLUDING MILO)	0.000500	1.00	0.06	TDS
276	O	WHEAT-ROUGH	0.000500	1.00	0.06	TDS
277	O	WHEAT-GERM	0.000500	1.00	0.06	TDS
278	O	WHEAT-BRAN	0.000500	1.00	0.06	TDS
279	O	WHEAT-FLOUR	0.000500	1.00	0.06	TDS
282	B	BEET SUGAR	0.000500	1.00	0.06	TDS
283	A	CANE SUGAR	0.000500	1.00	0.06	TDS
286	O	BUCKWHEAT	0.000500	1.00	0.06	TDS
289	O	CORN GRAIN-OIL	0.000002	1.00	0.06	FT
290	A	COTTONSEED-OIL	0.000480	1.00	0.06	FT
291	A	COTTONSEED-MEAL	0.024000	1.00	0.06	FT
292	A	FLAX SEED	0.032000	1.00	0.06	FTtr
293	A	PEANUTS-OIL	0.000380	1.00	0.06	FT
294	A	SAFFLOWER-SEED	0.024000	1.00	0.06	FTtr
295	A	SAFFLOWER-OIL	0.000450	1.00	0.06	FT

296	A	SESAME-OIL	0.001800	1.00	0.06	FT
297	G	SOYBEANS-OIL	0.000160	1.00	0.06	FT
298	A	SUNFLOWER-OIL	0.001800	1.00	0.06	FT
299	A	COCONUT-OIL	0.001800	1.00	0.06	FT
303	G	SOYBEANS-UNSPECIFIED	0.000500	1.00	0.06	TDS
304	G	SOYBEANS-MATURE SEEDS DRY	0.000500	1.00	0.06	TDS
309	A	SEEDS (PUMPKIN;etc)	0.000500	1.00	0.06	TDStr
318	X	MILK-NONFAT SOLIDS	0.000450	1.00	0.10	PDP
319	X	MILK-FAT SOLIDS	0.000450	1.00	0.10	PDP
320	X	MILK SUGAR (LACTOSE)	0.000450	1.00	0.10	PDP
321	U	BEEF-MEAT BYPRODUCTS	0.000680	1.00	0.22	PDPtr
322	U	BEEF(ORGAN MEATS)-OTHER	0.000680	1.00	0.22	PDPtr
323	U	BEEF-DRIED	0.000042	1.00	0.22	PDPtr
324	U	BEEF(BONELESS)-FAT	0.000007	1.00	0.22	PDPtr
325	U	BEEF(ORGAN MEATS)-KIDNEY	0.000240	1.00	0.22	PDPtr
326	U	BEEF(ORGAN MEATS)-LIVER	0.000680	1.00	0.22	PDPtr
327	U	BEEF(BONELESS)-LEAN (FAT/FREE)	0.000042	1.00	0.22	PDPtr
328	U	GOAT-MEAT BYPRODUCTS	0.000680	1.00	0.22	PDPtr
329	U	GOAT(ORGAN MEATS)-OTHER	0.000680	1.00	0.22	PDPtr
330	U	GOAT(BONELESS)-FAT	0.000007	1.00	0.22	PDPtr
331	U	GOAT(ORGAN MEATS)-KIDNEY	0.000240	1.00	0.22	PDPtr
332	U	GOAT(ORGAN MEATS)-LIVER	0.000680	1.00	0.22	PDPtr
333	U	GOAT(BONELESS)-LEAN (FAT/FREE)	0.000042	1.00	0.22	PDPtr
336	U	SHEEP-MEAT BYPRODUCTS	0.000680	1.00	0.22	PDPtr
337	U	SHEEP(ORGAN MEATS)-OTHER	0.000680	1.00	0.22	PDPtr
338	U	SHEEP(BONELESS)-FAT	0.000007	1.00	0.22	PDPtr
339	U	SHEEP(ORGAN MEATS)-KIDNEY	0.000240	1.00	0.22	PDPtr
340	U	SHEEP(ORGAN MEATS)-LIVER	0.000680	1.00	0.22	PDPtr
341	U	SHEEP(BONELESS)-LEAN (FAT FREE	0.000042	1.00	0.22	PDPtr
342	U	PORK-MEAT BYPRODUCTS	0.000680	1.00	0.22	PDPtr
343	U	PORK(ORGAN MEATS)-OTHER	0.000680	1.00	0.22	PDPtr
344	U	PORK(BONELESS)-FAT	0.000007	1.00	0.22	PDPtr
345	U	PORK(ORGAN MEATS)-KIDNEY	0.000240	1.00	0.22	PDPtr
346	U	PORK(ORGAN MEATS)-LIVER	0.000680	1.00	0.22	PDPtr
347	U	PORK(BONELESS)-LEAN (FAT FREE)	0.000042	1.00	0.22	PDPtr
354	W	FISH-FINFISH-SALTWATER-DRIED	0.500000	1.60	0.06	tolerance
355	V	TURKEY-BYPRODUCTS	0.006000	1.00	0.10	FT
356	V	TURKEY-GIBLETS (LIVER)	0.006000	1.00	0.10	FT
357	V	TURKEY-(BONELESS)-FAT	0.006000	1.00	0.10	FT
358	V	TURKEY-(BONELESS)LEAN/FAT FREE	0.006000	1.00	0.10	FT
359	V	TURKEY-UNSPECIFIED	0.006000	1.00	0.10	FT
360	V	POULTRY-OTHER-LEAN (FAT FREE)	0.006000	1.00	0.10	FT
361	V	POULTRY-OTHER-GIBLETS(LIVER)	0.006000	1.00	0.10	FT
362	V	POULTRY-OTHER-FAT	0.006000	1.00	0.10	FT
363	X	EGGS-WHOLE	0.000500	1.00	0.05	TDS
364	X	EGGS-WHITE ONLY	0.000500	1.00	0.05	TDS
365	X	EGGS-YOLK ONLY	0.000500	1.00	0.05	TDS
366	V	CHICKEN-BYPRODUCTS	0.006000	1.00	0.10	FT
367	V	CHICKEN-GIBLETS(LIVER)	0.006000	1.00	0.10	FT
368	V	CHICKEN (BONELESS)-FAT	0.006000	1.00	0.10	FT
369	V	CHICKEN(BONELESS)LEAN/FAT FREE	0.006000	1.00	0.10	FT
385	V	CHICKEN-GIBLETS (EXCL. LIVER)	0.006000	1.00	0.10	FT
399	O	OATS-BRAN	0.000500	1.00	0.06	TDS
408	O	RICE-BRAN	0.000500	1.00	0.06	TDS
417	A	SUNFLOWER-SEEDS-HULLED	0.000500	1.00	0.06	TDS
449	V	TURKEY-(ORGAN MEATS)-OTHER	0.006000	1.00	0.10	FT

Chronic Exposure (EX1) Analysis for Dichlorvos
RESIDUE FILE NAME: DDVP1PCT ANALYSIS DATE: 7-14-1998
NFCS Combined 89-92 DATA
DPR NOEL (Chronic) = 0.050000 mg/kg body-wt/day
COMMENT 1: All uses based on USEPA DRES (6/11/98)
COMMENT 2: USEPA residue and percent crop treated

TOTAL EXPOSURE BY POPULATION SUBGROUP

POPULATION SUBGROUP	TOTAL EXPOSURE mg/kg body wt/day	Percent of NOEL	Margin of Safety 1/
U.S. POP - 48 STATES - ALL SEASONS	0.000000757	0.0%	66,091
PACIFIC REGION	0.000000985	0.0%	50,770
HISPANICS	0.000000826	0.0%	60,538
NON-HISPANIC WHITES	0.000000736	0.0%	67,923
NON-HISPANIC BLACKS	0.000000833	0.0%	60,060
NON-HISPANIC OTHER THAN BLACK OR WHITE	0.000000783	0.0%	63,819
ALL INFANTS	0.000001557	0.0%	32,117
NURSING INFANTS (<1 YEAR OLD)	0.000000209	0.0%	238,919
NON-NURSING INFANTS (<1 YEAR OLD)	0.000002124	0.0%	23,541
CHILDREN (1-6 YEARS)	0.000001423	0.0%	35,131
CHILDREN (7-12 YEARS)	0.000000997	0.0%	50,172
FEMALES (13-19 YRS/NOT PREG. OR NURSING)	0.000000602	0.0%	83,057
FEMALES (20+ YEARS/NOT PREG. OR NURSING)	0.000000589	0.0%	84,886
FEMALES (13-50 YEARS)	0.000000574	0.0%	87,163
FEMALES (13+/PREGNANT/NOT NURSING)	0.000000552	0.0%	90,570
FEMALES (13+/NURSING)	0.000000780	0.0%	64,099
MALES (13-19 YEARS)	0.000000753	0.0%	66,365
MALES (20+ YEARS)	0.000000675	0.0%	74,112
SENIORS (55+)	0.000000730	0.0%	68,485

1. Margin of Safety = DPR NOEL / Dietary Exposure